

Presence of Asthma Risk Factors and Environmental Exposures Related to Upper Respiratory Infection–Triggered Wheezing in Middle School–Age Children

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Viral respiratory infections and exposure to environmental constituents such as tobacco smoke are known or suspected to trigger wheezing/asthma exacerbations in children. However, few population-based data exist that examine the relationship between wheezing triggered by viral respiratory infections and environmental exposures. In this investigation we used population-based data to evaluate differences in exposures between symptomatic middle school–age children who did and did not report wheezing triggered by viral respiratory infections. As part of the North Carolina School Asthma Survey (NCSAS), a 66-question data instrument was used to collect information from children enrolled in North Carolina public middle schools during the 1999–2000 school year. Associations between exposures and upper respiratory infection–triggered wheezing (URI-TW) among symptomatic children were examined using adjusted prevalence odds ratios (PORs). Video methods developed for the International Study of Asthma and Allergies in Childhood were used to assess wheezing. Among the 33,534 NCSAS symptomatic participants, positive associations were observed between most exposures and URI-TW. Reported presence of all allergy variables (PORs ranging from 2.11 to 2.45) was more strongly associated with URI-TW than either smoking or other exposures. Presence of URI-TW was higher at increasing levels of tobacco smoke exposure, but no apparent dose–response effect was observed for other indoor air pollutants. URI-TW in middle school children is most associated with reported allergen sensitivity, relative to other asthma risk factors and environmental exposures. Data from this investigation may be useful in developing assessment, screening, and targeting strategies to improve asthma and wheezing management in children. *Key words:* allergies, asthma, childhood, environment, exposures, infections, respiratory, rhinovirus, wheezing. *Environ Health Perspect* 111:657–662 (2003). doi:10.1289/ehp.5824 available via <http://dx.doi.org/> [Online 9 January 2003]

An estimated 5 million U.S. children per year suffer from asthma, the most common chronic childhood illness in the United States (AAANPO 2000). These numbers may underestimate the impact of the disease because childhood asthma is believed to be both widely underdiagnosed and undertreated (Pearce et al. 1998). Diagnosed asthma rates are highest in children compared with other age groups, and asthma is thought to have increased 92% in U.S. children over the past decade (AAANPO 2000).

Exacerbations of asthma are the major cause of morbidity and mortality in asthmatic children (Johnston 1998; Johnston et al. 1996). Clinical evidence suggests that viral infections, rather than bacterial infections, play the major role in asthma exacerbation caused by upper respiratory infections (URIs) (Abramson et al. 1995; Busse et al. 1997; Johnston 1998; Pearce et al. 1998; Weiss 1998). In addition, a study by Sarafino et al. (1998) examined the impact of various triggers for asthma symptoms at various ages and found that, of all triggers examined, the impact of respiratory infections declines the most with age in children. They concluded that as children get older, asthma episodes are more likely to be triggered by factors such as exercise and allergy problems.

Exposure to various constituents including tobacco smoke, airborne allergens, dust mites, mold, and other indoor air pollutants is known or suspected to trigger wheezing or asthmatic exacerbations in children (Pearce et al. 1998). Such exposures may have increasing importance to the lives of U.S. children, most of whom spend at least 90% of their time indoors (Bjorksten 1996). Despite this increased importance, few population-based data exist that examine whether exposure to environmental factors is different in symptomatic children whose wheezing is triggered by URIs (URI-TW) compared with children for whom URIs are not a trigger for their wheezing exacerbations.

The purpose of this investigation is to use population-based, self-reported data and internationally standardized methods from the International Study of Asthma and Allergies in Childhood (ISAAC) to evaluate whether differences in environmental exposures and asthma risk factors exist between currently symptomatic middle school–age children who continue to experience URI-TW and children for whom URIs are not a trigger. Using a large diverse population of symptomatic middle school children in North Carolina, a variety of exposures were

examined and compared between children who did and did not report URI-TW.

Materials and Methods

This work was completed as part of the North Carolina School Asthma Survey (NCSAS), a statewide surveillance project implemented by the North Carolina Department of Health and Human Services with assistance from the University of North Carolina (UNC) School of Public Health and in cooperation with the North Carolina Department of Public Instruction. Aims of NCSAS include *a*) describing the demographic distribution of asthma symptoms in North Carolina middle school–age children, *b*) assessing the magnitude of undiagnosed wheezing in the state of North Carolina, and *c*) identifying areas in the state that have the greatest burden from undiagnosed wheezing.

NCSAS used a 66-question data instrument to collect baseline prevalence data on asthmatic symptoms, risk factor and exposure information, functional consequences, and treatment outcomes from a target population of all middle school children (seventh and eighth grades) in North Carolina. The population was defined as being enrolled in seventh or eighth grade of a public school during the 1999–2000 school year. Active written informed consent for NCSAS participants was waived by the North Carolina Secretary of Health and Human Services because the purpose of NCSAS was collection of data for surveillance purposes, no personal identifiers were collected and retained, and there was no risk to the participants in participation. After the waiver of active written consent was obtained, the NCSAS investigation team submitted an application to the UNC School of Public Health institutional review board (IRB). The assistance provided to the North Carolina Department of Health and Human

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Services by UNC investigators in conducting the NCSAS was subsequently approved by the UNC IRB. Participation in NCSAS did not occur until written informed consent was waived by the North Carolina Secretary of Health and Human Services and after UNC IRB approval.

In December 1999, NCSAS questionnaires and question-by-question videotapes were distributed to principals of all North Carolina public schools containing middle school children, and data collection continued until June 2000. Participants self-completed the NCSAS data collection instrument at school while a question-by-question videotape was played. Standardized AVQ3 video methods and questions developed for ISAAC to ascertain presence of asthmatic symptoms in respondents were incorporated into the videotape (ISAAC Steering Committee 1998). Specifically, four visual displays for wheezing at rest, exercise-induced wheezing, nocturnal wheeze, and severe wheeze were shown, followed by standardized written questions in the questionnaire. A separate question asked whether children experienced wheezing with a head cold (i.e., URI-TW) during the previous 12 months.

Information on a variety of asthma risk factors and environmental exposures was collected in the NCSAS questionnaire, including parental history of asthma, presence of allergies, cigarette smoke exposure, use of plastic bed coverings (proxy for dust mite exposure reduction), presence of wall-to-wall carpeting, and exposure to pets and indoor air pollutants. Regarding exposure to indoor air pollutants, information on the frequency of use of natural gas kitchen stove, kerosene heater, wood-burning stove, and wood-burning fireplace was collected. Smoking information collected included whether the respondent ever smoked cigarettes, how many days and number of cigarettes smoked during the month before questionnaire completion, number of other people in the respondent's household who smoked, and how often the respondent was near enough to smell the smoke from other people's cigarettes.

Completed NCSAS questionnaires were returned and electronically scanned into a text file. Data analyses were performed using SAS version 8.2 (SAS Institute, Cary, NC). Only symptomatic children—defined as experiencing at least one episode of wheezing during the past year—were included in analyses. Wheezing episodes were determined by a positive response to any of the four core ISAAC video questions pertaining to wheezing in the NCSAS questionnaire (wheezing at rest, exercise-induced wheezing, nocturnal wheeze, or severe wheeze). Bivariate analyses were performed for each exposure variable versus the dichotomous

outcome of URI-TW to obtain unadjusted prevalence odds ratios (PORs) and 95% confidence intervals (CIs). Bivariate analyses were also performed for URI-TW and for several potential confounders, including sex, race/ethnicity, urban/rural residence, and socioeconomic status (SES). SES was determined by participation in a free/reduced school lunch program.

Stratified analyses and unconditional logistic regression models containing interaction terms were used to assess potential interaction. Absolute difference in stratum-specific odds ratios (ORs), absence of overlap between stratum-specific confidence intervals, Breslow-Day statistics, and *p*-values obtained from likelihood ratio tests were calculated and

used to determine statistical interaction between exposure and potential confounding variables (sex, race/ethnicity, urban/rural residence, SES, and recent smoking). Recent smoking or whether or not a participant reported smoking cigarettes during the 30 days before the questionnaire was completed classified as an exposure in the initial analysis and was assessed as a potential confounding variable and effect modifier for all analyses involving non-active-smoking variables. Confounding was addressed using unconditional logistic regression modeling. Adjusted PORs and 95% CIs were obtained from models containing the selected exposure variable, potential confounding variables, and any necessary interaction terms.

Table 1. Unadjusted PORs for the association of URI-TW with asthma risk factor and environmental exposures among NCSAS children reporting current wheezing.

Asthma risk factor/environmental exposure variable	URI-TW		Unadjusted POR (95% CI)
	Yes (n)	No (n)	
Maternal asthma history			
Yes	2,971	1,587	1.80 (1.68–1.93)
No	6,650	6,392	
Paternal asthma history			
Yes	2,144	1,050	2.00 (1.84–2.17)
No	5,590	5,473	
Allergies—presence of symptoms			
Yes	13,365	9,525	2.07 (1.98–2.18)
No	3,883	5,742	
Dog allergies			
Yes	2,244	920	2.55 (2.35–2.77)
No	11,143	11,661	
Cat allergies			
Yes	4,062	1,847	2.48 (2.33–2.63)
No	9,547	10,754	
Dust allergies			
Yes	10,075	5,733	2.79 (2.66–2.94)
No	4,095	6,511	
Grass/pollen allergies			
Yes	10,156	6,110	2.55 (2.43–2.69)
No	4,132	6,351	
Ever smoked cigarettes			
Yes	7,615	5,814	1.31 (1.25–1.37)
No	9,455	9,470	
No. days smoked in past 30 days			
0	3,194	2,952	1.00
1–7	2,006	1,421	1.30 (1.20–1.42)
8–20	652	443	1.36 (1.19–1.55)
Almost every day	1,553	835	1.72 (1.56–1.90)
No. cigarettes smoked per day—past 30 days			
Did not smoke	3,164	2,916	1.00
1	1,442	958	1.38 (1.26–1.53)
2–10	1,669	1,111	1.38 (1.26–1.52)
> 10	700	330	1.95 (1.70–2.25)
How many other people in household smoke cigarettes			
0	6,331	6,595	1.00
1	4,945	4,335	1.19 (1.13–1.25)
2	3,673	2,939	1.30 (1.23–1.38)
≥ 3	2,143	1,441	1.55 (1.44–1.67)
Near enough to smell or breathe smoke from other people's cigarettes			
Never	1,397	1,965	1.00
Less than once per week	2,198	2,507	1.23 (1.13–1.35)
Once per week	1,534	1,469	1.47 (1.33–1.62)
2–4 times per week	2,428	2,069	1.65 (1.51–1.81)
Nearly every day	9,567	7,311	1.84 (1.71–1.98)

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Results

School response rate for the NCSAS was 88% of the 565 eligible schools in 99 of 100 counties. The NCSAS participants ($n = 128,568$) encompassed barely more than two-thirds of the approximately 192,000 North Carolina public school children enrolled in the seventh and eighth grade during the 1999–2000 school year (North Carolina State Department of Public Instruction. Unpublished data). No differences were found when demographic and socioeconomic characteristics of students from schools that participated and did not participate in NCSAS were compared (Yeatts et al. In press).

Of the 128,568 children in the NCSAS study population, 33,534 reported experiencing

an episode of wheezing in the previous year. Among these currently symptomatic children, 17,358 (52%) reported experiencing at least one episode of wheezing triggered by a head cold during the previous year (URI-TW), 15,502 (46%) reported no URI-TW, and data were missing for 674 (2%) of children. Also, among the 33,534 children, 55% were female, 95% were 12–14 years old, 38% participated in free/reduced school lunch programs (used as a marker for lower SES), and 52% were from urban areas. Sixty percent were white, 29% were African American, and 11% identified themselves as another race/ethnicity.

Bivariate analysis of risk factors and environmental exposures with URI-TW revealed positive associations between most exposures

and URI-TW (Table 1). Reported presence of allergies (PORs ranging between 2.07 and 2.79) was more strongly associated with URI-TW than either smoking or exposure to other indoor air pollutants (PORs < 2.00 for all levels of all exposures). URI-TW increased at increasing exposure to smoking. No apparent dose–response effect was observed for the other indoor air pollutants or exposures except for wall-to-wall carpeting throughout the house. Parental history of asthma was moderately associated with URI-TW, with reported paternal history showing a slightly higher association than maternal history (POR = 2.00 vs. POR = 1.80, respectively). Interestingly, reported use of plastic bed coverings was positively associated with URI-TW (POR = 1.32; 95% CI, 1.23–1.41).

Bivariate analyses also indicated that females had a slightly higher prevalence of URI-TW than did males (POR = 1.54; 95% CI, 1.48–1.61), and lower SES and rural residence were only slightly associated with the URI-TW (POR = 1.05 and 1.15, respectively) (Table 2). Race/ethnicity was not strongly associated with URI-TW. Compared with white non-Hispanics, a small protective effect was observed for black/African American non-Hispanic and Asian/Pacific Islander non-Hispanic race ethnicities, and no effect was observed for children of Hispanic race/ethnicity. Slight positive associations were observed for Native American/Eskimo non-Hispanic and other race/ethnicities and URI-TW.

Stratified analyses and likelihood ratio test results indicated only one interaction: the association between second-hand smoke and URI-TW varied by recent smoking status. Recent nonsmokers were at greater risk for URI-TW at all levels of second-hand smoke exposure (PORs of 1.47, 1.65, 1.85, and 2.06 from lowest to highest exposure level) compared with smokers (PORs of 0.95, 0.99, 1.26, and 1.44). Adjusting for potential confounding variables using unconditional logistic regression modeling either slightly reduced or did not meaningfully change the effect of all exposure variables on the outcome of URI-TW (Table 3). After adjusting for confounding, most exposures remained positively associated with the outcome, with allergy-related exposures remaining most strongly associated with URI-TW. In addition, the same trends observed in the bivariate and stratified analyses for smoking-related exposures were observed in the adjusted analyses.

Discussion

Results from this investigation indicate that URI-TW in middle school children is most associated with reported allergen sensitivity, relative to other asthma risk factors and environmental exposures. Reporting of all types of

Table 1. Continued.

Asthma risk factor/environmental exposure variable	URI-TW		Unadjusted POR (95% CI)
	Yes (n)	No (n)	
Dog exposure during past 12 months			
Never	945	1,135	1.00
Less than once per month	1,036	974	1.28 (1.13–1.44)
1–3 times per month	1,052	982	1.29 (1.14–1.45)
About once per week	1,690	1,545	1.31 (1.18–1.47)
Almost every day	12,149	10,425	1.40 (1.28–1.53)
Cat exposure during past 12 months			
Never	3,498	3,657	1.00
Less than once per month	2,047	1,939	1.10 (1.02–1.19)
1–3 times per month	1,455	1,294	1.18 (1.08–1.28)
About once per week	2,050	1,642	1.31 (1.21–1.41)
Almost every day	7,324	6,062	1.26 (1.19–1.34)
Gas stove use during an average month			
Never	11,692	10,993	1.00
Less than once per month	534	346	1.45 (1.26–1.67)
1–3 times per month	538	379	1.33 (1.17–1.52)
About once per week	763	693	1.04 (0.93–1.15)
Almost every day	3,494	2,740	1.20 (1.13–1.27)
Kerosene heater use in cold weather ^a			
Never	11,104	10,415	1.00
Less than once per month	1,146	868	1.24 (1.13–1.36)
1–3 times per month	698	524	1.25 (1.11–1.40)
About once per week	747	647	1.08 (0.97–1.21)
Almost every day	3,004	2,472	1.14 (1.07–1.21)
Wood-burning stove use in cold weather ^a			
Never	14,252	13,127	1.00
Less than once per month	360	254	1.31 (1.11–1.54)
1–3 times per month	262	181	1.33 (1.10–1.61)
About once per week	267	200	1.23 (1.02–1.48)
Almost every day	1,005	685	1.35 (1.22–1.49)
Wood fireplace use in cold weather ^a			
Never	10,959	10,376	1.00
Less than once per month	1,225	982	1.18 (1.08–1.29)
1–3 times per month	982	846	1.10 (1.00–1.21)
About once per week	1,050	923	1.08 (0.98–1.18)
Almost every day	2,174	1,506	1.37 (1.27–1.47)
Mold/mildew in home ^a			
Yes	1,186	677	1.62 (1.47–1.78)
No	14,301	13,195	
Plastic bed coverings			
Yes	2,639	1,804	1.32 (1.23–1.41)
No	13,603	12,583	
Wall-to-wall carpeting in household			
None	2,310	2,548	1.00
Some of the house	4,285	3,789	1.24 (1.16–1.34)
Most of the house	9,778	8,001	1.35 (1.26–1.44)
Wall-to-wall carpeting in room where sleep			
Yes	12,178	10,230	1.17 (1.11–1.23)
No	3,898	3,838	

^aDuring the 12 months before questionnaire completion.

allergies, including dog, cat, dust, and grass/pollen, as well as the presence of allergic symptoms was consistently greater among children who reported wheezing with a head cold during the previous year compared with children who did not report URI-TW. Although it is not surprising that allergen sensitivity was high among the symptomatic study participants, because approximately 80% of diagnosed asthmatics are sensitized to at least one common airborne allergen (Pearce et al. 1998), the consistently higher prevalence of allergen reporting among the children with URI-TW is significant. These data indicate that allergen-sensitive children at middle school age, independent of asthma diagnosis, are more susceptible to the effects of head colds with regard to triggering their wheezing symptoms.

These population-based findings serve to analytically validate the relationship between allergen sensitivity and URI acquisition in producing an exacerbation of wheezing in children. Gern and Busse (1999) previously described such an association, suggesting that acute viral infections in the presence of allergen sensitivity may have a synergistic effect on lower airway physiology that greatly increases inflammation and likelihood of wheezing. A study by Duff et al. (1993) provides evidence to support such a synergistic effect. Using infant and child admission data from a hospital emergency department, they concluded the odds of having respiratory allergies (OR = 4.5) or a viral infection (OR = 3.7) were higher in children 2 years or older with wheezing than in children without wheezing (Duff et al. 1993). In addition, Duff et al. (1993) found that the odds of having both respiratory allergies and a viral infection in presenting at the emergency room were 10-fold higher (OR = 10.8) in the wheezing children versus the children without wheezing.

It has been postulated that eliciting immune reactivity in the lower airway, specifically the release of proinflammatory cytokines and mediators, is the mechanism by which viral respiratory infections trigger exacerbations of asthma symptoms (Gern and Busse 1999; Grunberg and Sterk 1999). Although critical to clear virus from the airway, such an immune response may also lead to airway obstruction and respiratory symptoms, particularly in children with preexisting airway inflammation, such as those susceptible to airborne allergens (Gern and Busse 1999). The infection may also extend to the lower airway when an exacerbation occurs (Gern and Busse 1999). In addition, experimental evidence indicates that such an effect may last for weeks after acute infection (Grunberg and Sterk 1999). Furthermore, a study by Marin et al. (2000) indicated that URI-associated viruses may behave more pervasively and persistently

Table 2. PORs for the association of URI-TW with demographic variables among NCSAS children reporting current wheezing.

Demographic variable	URI-TW		Unadjusted POR (95% CI)
	Yes (n)	No (n)	
Sex			
Female	10,303	7,562	1.54 (1.48–1.61)
Male	6,860	7,771	
Race/ethnicity			
White ^a	9,846	8,621	1.00
Black/African American ^a	4,445	4,268	0.91 (0.87–0.96)
Hispanic	1,544	1,347	1.00 (0.93–1.09)
Native American/Eskimo ^a	371	254	1.28 (1.09–1.50)
Asian/Pacific Islander ^a	153	233	0.57 (0.47–0.71)
Other	848	651	1.14 (1.03–1.27)
SES			
Low	6,493	5,636	1.05 (1.00–1.10)
High	10,429	9,474	
Urban/rural residence			
Rural	9,329	7,794	1.15 (1.10–1.20)
Urban	8,029	7,708	

^aNon-Hispanic.

Table 3. Comparison of adjusted and unadjusted PORs for the association of URI-TW with asthma risk factor and environmental exposure variables among NCSAS children reporting current wheezing.

Asthma risk factor/environmental exposure variable	Unadjusted POR (95% CI)	Adjusted POR (95% CI)
Maternal asthma history	1.80 (1.68–1.93)	1.58 (1.41–1.77)
Paternal asthma history	2.00 (1.84–2.17)	1.94 (1.69–2.23)
Allergies—presence of symptoms	2.07 (1.98–2.18)	2.11 (1.94–2.29)
Dog allergies	2.55 (2.35–2.77)	2.29 (1.99–2.64)
Cat allergies	2.48 (2.33–2.63)	2.28 (2.05–2.54)
Dust allergies	2.79 (2.66–2.94)	2.45 (2.25–2.66)
Grass/pollen allergies	2.55 (2.43–2.69)	2.33 (2.14–2.54)
Ever smoked cigarettes	1.31 (1.25–1.37)	1.29 (1.23–1.35)
Days smoked in past 30 days		
0	1.00	1.00
1–7	1.30 (1.20–1.42)	1.31 (1.20–1.43)
8–20	1.36 (1.19–1.55)	1.38 (1.21–1.58)
Almost every day	1.72 (1.56–1.90)	1.67 (1.50–1.85)
Cigarettes smoked per day—past 30 days		
Did not smoke	1.00	1.00
1	1.38 (1.26–1.53)	1.38 (1.25–1.52)
2–10	1.38 (1.26–1.52)	1.37 (1.24–1.50)
> 10	1.95 (1.70–2.25)	1.93 (1.66–2.24)
How many other people in household smoke cigarettes		
0	1.00	1.00
1	1.19 (1.13–1.25)	1.18 (1.11–1.24)
2	1.30 (1.23–1.38)	1.26 (1.19–1.34)
≥ 3	1.55 (1.44–1.67)	1.50 (1.39–1.62)
Near enough to smell or breathe smoke from other people's cigarettes (recently smoked = yes)		
Never	1.00	1.00
Less than once per week	1.23 (1.13–1.35)	0.97 (0.69–1.36)
Once per week	1.47 (1.33–1.62)	0.97 (0.69–1.36)
2–4 times per week	1.65 (1.51–1.81)	1.29 (0.96–1.74)
Nearly every day	1.84 (1.71–1.98)	1.41 (1.08–1.84)
Near enough to smell or breathe smoke from other people's cigarettes (recently smoked = no)		
Never	1.00	1.00
Less than once per week	1.23 (1.13–1.35)	1.38 (1.06–1.78)
Once per week	1.47 (1.33–1.62)	1.64 (1.24–2.16)
2–4 times per week	1.65 (1.51–1.81)	1.77 (1.38–2.28)
Nearly every day	1.84 (1.71–1.98)	1.92 (1.55–2.39)
Dog exposure during past 12 months		
Never	1.00	1.00
Less than once per month	1.28 (1.13–1.44)	1.15 (0.92–1.45)
1–3 times per month	1.29 (1.14–1.45)	1.41 (1.12–1.76)
About once per week	1.31 (1.18–1.47)	1.26 (1.03–1.54)
Almost every day	1.40 (1.28–1.53)	1.25 (1.05–1.48)

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in asthmatics, documenting a high degree of persistence of genetic material from common URI-associated viruses in the nasal discharges of asthmatics who had been symptom-free for at least 3 weeks, compared with almost no persistence among nonasthmatic controls.

In contrast to allergies, neither active nor passive cigarette smoke exposure was strongly associated with URI-TW. Little association was observed between recent active smoking and presence of at least one allergy (OR = 1.13; 95% CI, 1.02–1.25). Nonetheless, an increased positive association with URI-TW was observed with both active and passive smoke as smoke exposure increased, indicating that children exposed to greater levels of cigarette smoke display a greater prevalence of wheezing triggered by head colds. In addition, the association between passive smoking and URI-TW was more pronounced among non-smoking children compared with children who reported recently smoking cigarettes. Increased exacerbations of asthma have been observed in children exposed to passive smoking (Abramson et al. 1995; Sears 1997), and

our data suggest that children who do not themselves smoke may be at a slightly greater risk of an acute exacerbation when exposed to both passive tobacco smoke and infectious agents that cause URIs.

Compared with children who do not report wheezing with head colds, middle school-age children experiencing URI-TW reported only slightly greater exposure to indoor air pollutants from gas, kerosene, and wood burning sources, as well as to pets and wall-to-wall carpeting. These data suggest that there is little association between the presence of indoor air pollutants and whether or not a child of middle school age experienced wheezing with a head cold. However, our exposure data, especially regarding indoor heating and cooking devices, are limited by self-report; no measure of actual exposure to indoor air pollutants was taken in this investigation. One additional finding was that use of plastic bed coverings showed a slight positive association with URI-TW. This trend may be due to a greater inherent susceptibility to exacerbation of symptoms from a variety of triggers,

including URIs, among children who use plastic bed coverings to reduce the effects of dust mite exposure.

We used the ISAAC video questionnaire to determine our study population of children reporting current wheezing from the overall NCSAS study population. The ISAAC video questionnaire was developed with the premise that showing rather than describing clinical asthma would produce greater validity and repeatability and would achieve a more accurate recognition of clinical asthma (Beasley et al. 1998; Crane et al. 1995). Written questionnaires using the term “diagnosed asthma” have poor sensitivity in that a number of children with asthma would not have received a prior diagnosis (Beasley et al. 1998). Four studies have compared the validity of the ISAAC video questions with comparable questions from written questionnaires in determining asthma in children 13–16 years old, and all determined that the ISAAC video questionnaire was effective in determining bronchial hyperresponsiveness and asthma prevalence in children 13–16 years old (Gibson et al. 2000; Lai et al. 1997; Shaw et al. 1992, 1995).

Inferences based on the results of analyses conducted using NCSAS data are limited to the point of time of questionnaire administration because of the cross-sectional study design of NCSAS. URI data were limited to the respondent's interpretation of a head cold, leading to potential misclassification if respondents included other similar conditions such as allergic rhinitis. Also, we did not determine the frequency of head colds during the past year, and it is possible that some respondents who would have experienced wheezing with a URI in the 12 months before questionnaire administration did not experience a head cold during that period of time. However, with high frequency of URI transmission among schoolchildren and because URI incidence has varied little in the past 60 years (Graham 1990), the number of respondents with wheezing who did not experience a head cold in the previous year is presumed to be low. Reporting of exposures was limited to self-report; no medical histories (allergies, parental history of asthma) or pollutant measurements were taken. Therefore, some of the findings, especially regarding indoor air pollutants, must be interpreted cautiously.

In this investigation we used comprehensive surveillance data collected from a large population of children from a geographically and demographically diverse setting. Our analyses investigating head colds as triggers for wheezing included all children who reported experiencing wheezing and were not limited to children who have been diagnosed with asthma in a clinical setting. Data used in these analyses were collected firsthand from the respondents themselves, instead of relying on

Table 3. *Continued.*

Asthma risk factor/environmental exposure variable	Unadjusted POR (95% CI)	Adjusted POR (95% CI)
Cat exposure during past 12 months		
Never	1.00	1.00
Less than once per month	1.10 (1.02–1.19)	1.12 (0.98–1.28)
1–3 times per month	1.18 (1.08–1.28)	1.23 (1.05–1.43)
About once per week	1.31 (1.21–1.41)	1.30 (1.14–1.49)
Almost every day 1.26 (1.19–1.34)	1.23 (1.11–1.36)	
Gas stove use during an average month		
Never	1.00	1.00
Less than once per month	1.45 (1.26–1.67)	1.26 (1.01–1.59)
1–3 times per month	1.33 (1.17–1.52)	1.49 (1.20–1.85)
About once per week	1.04 (0.93–1.15)	1.12 (0.93–1.35)
Almost every day 1.20 (1.13–1.27)	1.24 (1.13–1.36)	
Kerosene heater use in cold weather ^a		
Never	1.00	1.00
Less than once per month	1.24 (1.13–1.36)	1.16 (0.99–1.34)
1–3 times per month	1.25 (1.11–1.40)	1.38 (1.14–1.68)
About once per week	1.08 (0.97–1.21)	1.21 (1.01–1.45)
Almost every day 1.14 (1.07–1.21)	1.13 (1.03–1.24)	
Wood-burning stove use in cold weather ^a		
Never	1.00	1.00
Less than once per month	1.31 (1.11–1.54)	1.48 (1.13–1.94)
1–3 times per month	1.33 (1.10–1.61)	1.55 (1.14–2.11)
About once per week	1.23 (1.02–1.48)	1.29 (0.96–1.74)
Almost every day 1.35 (1.22–1.49)	1.39 (1.19–1.62)	
Wood fireplace use in cold weather ^a		
Never	1.00	1.00
Less than once per month	1.18 (1.08–1.29)	1.28 (1.09–1.49)
1–3 times per month	1.10 (1.00–1.21)	1.10 (0.94–1.30)
About once per week	1.08 (0.98–1.18)	1.18 (1.01–1.39)
Almost every day	1.37 (1.27–1.47)	1.39 (1.24–1.56)
Mold/mildew in home ^a	1.62 (1.47–1.78)	1.72 (1.48–2.01)
Plastic bed coverings	1.32 (1.23–1.41)	1.33 (1.20–1.49)
Wall-to-wall carpeting in household		
None	1.00	1.00
Some of the house	1.24 (1.16–1.34)	1.28 (1.13–1.44)
Most of the house	1.35 (1.26–1.44)	1.41 (1.27–1.57)
Wall-to-wall carpeting in room where sleep ^a	1.17 (1.11–1.23)	1.24 (1.14–1.36)

Adjusted PORs and 95% CIs were obtained from unconditional logistic regression modeling controlling for sex, race/ethnicity, SES, urban/rural residence, and, except for active smoking exposure variables, recent active smoking.

^aDuring the 12 months before questionnaire completion.

secondhand sources such as medical records or parental interviews, the latter of which can be a less reliable source of information than the information collected upon directly interviewing children of this age group (Braun-Fahrlander et al. 1998; Sole et al. 1998). We were able to collect information, analyze data, and report results on a variety of asthma risk factors and environmental exposures regarding the outcome of URI-TW while controlling for several potential confounding factors, including race/ethnicity, SES, urban-rural residence, and cigarette smoking.

To our knowledge, this is the first large population-based study to examine associations among environmental exposures, asthma risk factors, and wheezing triggered by URIs. The results from this study indicate that presence of allergies has the greatest association with wheezing triggered by head colds, relative to other asthma risk factors and environmental constituents, including cigarette smoke. These population-based data validate anecdotal, experimental, and clinical evidence suggesting an increased risk of wheezing among children with allergies who contract URIs. At the same time, we found little evidence for a substantial relationship between URI-TW and other environmental exposures, including cigarette smoke. Although more research is needed to supplement the findings of this investigation, these data may be useful in helping to develop assessment, screening, and targeting strategies to improve asthma and wheezing management in children. Development of such strategies may be of

particular importance to allergic children, who may be most at risk from severe consequences due to asthma symptoms occurring with head colds.

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